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EDITORIAL.

A RECENTLY reported fatality points once more to the moral that it is unwise to experiment upon oneself with anæsthetics unless there is a second person present. It is not many years since S. R. Wilson, one of the best-known anæsthetists in the North of England and a founder of this Journal, lost his life in exactly the same manner. It is natural for the conscientious anæsthetist to wish to know by personal experience exactly what it is that he presents to the patient who inhales an anæsthetic. Indeed, there is almost a tradition in this self-experimenting, handed on from the days when Simpson and his family and friends fell about the family dining-table in various degrees of chloroform narcosis.

In those comparatively crude times, however, the full dangers of anæsthesia were not yet understood. To-day we know enough to make every anæsthetist aware that he is taking an undue risk when he inhales any narcotic without the presence of another individual. Even the comparatively safe nitrous oxide becomes dangerous when it is possible for the administrator, who is also the inhalant, to become more firmly fixed into the face-piece when he is rendered unconscious, and thus to asphyxiate himself at the very moment when more air or oxygen is required.

It is difficult to believe that the results of self-experiment justify the risks. On the face of it, it is plain that only subjective sensations can be investigated or recorded, and of these only those arising early in the inhalation or late during recovery are likely to be trustworthy. The very nature of general anæsthetics makes them the last things with which

auto-experiment can usefully be performed. There would be far more justification for testing local analgesia by cutting oneself about; that, at any rate, could be carried out with a clear brain. The object of experiment is accurate observation. Accurate observation requires a clear brain. How, then, can self-experiment be successful when it is carried out on substances the prime function of which is to confuse cerebration?

SUB-ARACHNOID BLOCK; GENERAL ANALGESIA; "SPINAL" ANÆSTHESIA; RESPIRATORY PARALYSIS; FALLACIES AND METHODS.*

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THE title of this paper comprehends all the results obtainable on the nervous system by the injection of analgesic substances within the sub-arachnoid space. It includes nothing which was not observed as a result of the earliest employment of cocaine, but the interpretation of the results over the intervening period has been clouded with error, copied from one textbook or paper into another, and persisting in some of the latest publications. The basis of understanding is anatomical and physical.

Anatomy and Physics.

"The arachnoid membrane is a gauzy reticulum of almost web-like delicacy which in reality pervades the space it occupies. Its outer surface, or that closely related to the dura mater and bounding the sub-dural cavity, alone shows a sufficiently organised structure to merit the name of a membrane. This quickly grades into a loose, spongy reticulum which pervades the thick sub-arachnoid cavity throughout, and the strands of which are directly continuous into the more compact tissue of the pia mater. This sponge-like arachnoid tissue holds the cerebro-spinal fluid of the sub-arachnoid cavity, the meshes of the sponge constituting a reticular web of inter-communicating spaces. The cranial

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sub-arachnoid cavity is larger and the strands of the web are relatively more abundant than in the spinal canal." (Morris.)

The spinal sub-arachnoid cavity passes up through the foramen magnum and enlarges into the cisterna magna which is posterior, and in the dorsal decubitus dependent, and into the cisterna pontis and the cisterna basalis which are anterior, more elevated, and contain the cranial nerve roots. The sub-arachnoid space surrounds the entire brain stem, and the roots of all the nerves in the body pass through it. In this situation they are bare of fibrous sheath, and therefore easily affected by analgesic solutions. The sensory root of the fifth cranial is the largest and most compact in the body, but the spinal roots have their constituent fibres spread out into a fan-like arrangement of entry and exit from the cord. Those of the cervical and lumbar enlargements supplying the limbs are more bulky than those of the intervening thoracic nerves. The lowest roots of the conus medullaris are centrally placed in the cauda equina and are overlaid by the higher trunks of the leash. As these higher lumbar nerves escape through their foramina, the lower become more isolated and exposed. The pia mater spinalis consists of two layers, the outer fibrous, and between them ramifies a rich plexus of blood vessels. The dentate ligaments support the cord in its central position, but do not effectively divide the space into an anterior and posterior compartment. The reticulate formation of the arachnoid, the structure of the pia mater, and the vascularity of the intra-dural contents, the superficial area of which is relatively very great, are factors of prime importance which determine the distribution of soluble injected substances.

The cerebro-spinal[1] fluid is secreted by the chorioid plexuses, and passes through the medial and lateral foramina of the fourth ventricle into the cisterna magna, whence it is distributed over the brain and cord. The secretion of fluid can be very rapid, and it is probable that when a large quantity is withdrawn it is rapidly replaced. The absorption of additional fluid injected is also very rapid, and because of the great vascularity of the lining membranes and the large surface area, the absorption of foreign chemical

substances into the blood is only second in effectiveness and rapidity to direct intravenous injection, the rate of absorption being proportional to the concentration, and decreasing progressively as this becomes lowered. Though it is impossible to imagine complete stasis in any living body fluid, there is not in the spinal canal any movement which can be called a circulation which would effect the immediate distribution of drugs injected. Waves of condensation and rarefaction are communicated to the fluid by the respiratory and cardiac impulses, which may have some effect over a more prolonged period. The specific gravity of the fluid is not constant, but varies between 1.004 and 1.010. The movement of injected solutions is determined by their relative weight. The sinking of a hyperbaric fluid in one which is relatively hypobaric is determined by a difference so delicate that an estimation of the difference would be impossible by any coarse method. If the surface layer of water is cooled it sinks to the bottom of the container, or if the bottom layer is warmed it rises to the top. Thus a hypobaric injection moves only when the heavier cerebro-spinal fluid can descend from above and displace it upward. The rate of movement will depend on the degree of difference between the specific gravities of the two fluids and the angle which the spine makes with the horizontal. The behaviour of solutions injected into the sub-arachnoid space is modified by the anatomical contents and the absorptive surface to which they are exposed.

Fallacies.

The experimental work of Corning and its clinical application by Bier and other subsequent workers is described in most textbooks on the subject, but it is noteworthy that operations on the head and neck were performed under this method early in its history, long before Barker experimented in this country. This is not surprising, since the moderate Trendelenburg position has always been thought necessary to ensure a good blood supply to the medullary centres, and some of the solutions then used were among the heaviest ever employed, while cocaine was used in dosage not thought permissible to-day. It was this extension of the

analgesia to the head, and a high mortality rate, which proved so alarming to the early workers, who had an abiding fear of the direct action of the drug on the medullary centres, and stimulated Barker[3] to try to devise a method whereby the effect could be controlled to the lower regions of the body. Barker demonstrated that the drug could not spread upward by a process of simple diffusion, *a fact which is indisputable*, and by injecting relatively heavy solutions into glass tubes shaped to the curve of the spine and containing saline solution, he showed that unless the inclination was too great the solution descended to the lowest part of the dorsal curve, and "here it would remain more or less undiluted in contact with the structures around."

These glass-tube experiments have been the origin of much misconception, because there is no clear run down for the solution in the body, nor do they represent the rapid absorption by the tissues and into the blood which is such an important feature *in vivo*. They also gave the impression that the dose descended in concentrated form to a certain level of the cord, where, if held long enough, it became "fixed" by the nerve tissue. Barker refers to the action of the drug on the roots, but writes more frequently of the cord, suggesting that he was not clear on this point. The term "fixed" is very inapt, because a very large portion of whatever drug is injected is rushing round the body in the circulation until it is either excreted or destroyed. Remembering that before Barker's time the saline and other solutions were said to "diffuse" into the neck, and that analgesia of the whole body was obtained, it is not surprising to find that his heavy stovain glucose solution, S.G. 1.030, controlled by dosage and posture to the dorsal region, came to be known as "indiffusible" and the saline solutions as "diffusible."

This in spite of the fact that the "diffusible" Chaput solution, S.G. 1.080, was heavier than anything Barker subsequently employed. Arising from this muddle is the fallacy prevalent in our textbooks to-day that mixtures containing glucose are heavy solutions and saline solutions "light" solutions. Gwathmey[3] states that "solutions of lighter specific gravity than the cerebro-spinal fluid are

generally called diffusible, whereas those of heavier specific gravity are called non-diffusible." Actually 10 per cent novocain in distilled water, S.G. 1.017, is a heavy solution. Another error prevalent in America and sponsored by Labat[4] is that when cerebro-spinal fluid is the solvent of novocain crystals no spread by gravity will take place in the Trendelenburg position. It is impossible to add anything soluble to the cerebro-spinal fluid without increasing the specific gravity of that portion of it which happens for the time being to be solvent. Assuming a fluid of S.G. 1.006, then a 10 per cent addition of novocain crystals will raise the S.G. to 1.023. Addition of 250 mg. to 10 c.c. of fluid will raise the S.G. to 1.010, and will operate in precisely the same manner as that amount of 2½ per cent novocain in normal saline and will be subject to gravitational diffusion. The large volume of the low concentration will move just as effectively, though at first more slowly, as the small volume of the high concentration.

Spinocain and Durocaine.

The addition of alcohol to a solution to make it lighter, attempted by Babcock in 1909 and patented by Dr. Pitkin nearly twenty years later, serves only to effect a temporary reduction of specific gravity after injection. The two mixtures, spinocain, S.G. 1.005, and durocaine, S.G. 1.002, appear to respond to a variety of different treatments. An expansion of the solution over the thoracic nerve roots is said to be effected by an addition of cerebro-spinal fluid of anything between 1 and 10 c.c. in volume without regard to the length of the spine. After injection, some tilt the table into the head-up position and others into the Trendelenburg. For the past two and a half years incomparable analgesias have been claimed in the dorsal decubitus, but recently both administrator and patient have made a *volte face*, and the ventral position is adopted. A viscous substance called gliadine, 0.00325 gram per c.c. is said to delay absorption and to prolong analgesia, but the dosage has coincidentally increased beyond anything previously heard of. One claims constant complete abdominal analgesia in the up-tilt lateral position in one minute twenty-five

seconds, another that it may be delayed as long as forty-seven minutes. These fall into a separate system known as "controllable spinal analgesia." One observer has asked, "After all, does controllability matter?"

The explanation of the fallacies underlying the administration of these solutions is a very simple one. The ingredients fall into two distinct classes, volatile and non-volatile. The low specific gravity is due entirely to the volatile member. The rate of absorption into the blood of a volatile substance is out of all proportion more rapid than that of a non-volatile substance. If spinocain were floated on to the surface of a warm saline, the alcohol would evaporate into the air, and the novocain would be dissolved in the saline. When injected into the sub-arachnoid space and mixed with cerebro-spinal fluid, the specific gravities of the two fluids become averaged and the mixture only slightly hypobaric. The rate of upward displacement would therefore be slow and progressively retarded by further mixture with heavier fluid. The "air bubble in a spirit level" arrangement is possible only between two media which are not miscible. The rate of absorption of the volatile alcohol will be far more rapid than the non-volatile novocain, and the solution quickly passes through an isobaric stage and becomes hyperbaric. The end result is always novocain dissolved in cerebro-spinal fluid. The one constant feature in the ritual of these administrations is the Trendelenburg position.

The dosage of novocain is so high that the amount absorbed into the blood causes a general analgesic state, and the itching, drowsiness, and stupidity of the patient which has been described by various observers[5] is evidence of the action of the circulating drug on the sensory nerve endings in the skin and on the cortical cells of the cerebrum. When the Trendelenburg position is adopted immediately after injection the solution hangs until the alcohol has been absorbed, when gravitational diffusion commences. All the novocain absorbed into the blood before this begins serves only to poison the patient and effects no useful purpose. In consequence, the dosage sometimes used is more than double

that which is necessary for the production of analgesia by a more rational method.

Gravitational Diffusion.

The process of diffusion by gravity in the body differs from the descent of a heavy fluid *in vitro*, because of the meshwork of baffles which the solution encounters, resulting in more and more mixture with the lighter fluid, and the fact that the drug is all the while being rapidly extracted into the blood and by the nerves. Thus absorption and dilution result in progressive diminution of specific gravity and a progressive slowing of the movement, which is also limited in the horizontal position by the curve of the spine. In the Trendelenburg position the effect of gravity is not to move the dose in high concentration to the most dependent part, but to spread it out in ever decreasing concentration over a distance which will be proportional to the amount of drug injected, and as more butter is required to spread a large piece of bread than a small one, so more drug is required to spread a long spine than a short one, a factor which has never been considered. The body weight is important in relation to blood dosage, a fact which is apparent when considering the high relative dosage and frequent development of sleep in children, reported from the earliest period to the present day.

The dentate ligaments no doubt have at first some directing action in supporting and spreading the solution over the anterior roots, but it always tends to sag and give the greater concentration to the posterior roots, especially in the higher regions. When the high tide of effective concentration reaches well above the centre, then both anterior and posterior roots receive the drug; when it sinks below the centre, then only posterior roots are affected. This result, together with the fact that with moderate dosage only a low concentration reaches the neck, explains how the motor roots of the phrenics and upper intercostals can carry on when the head is analgesic. Analgesia always extends higher than paralysis, and the upper segments of the abdominal muscles may be imperfectly relaxed with absence of pain sense at the same and at higher levels. The arms are not paralysed

even in the highest analgesias by this method. The graded effect of all analgesics on mixed nerves has long been known both experimentally and clinically. Pain sense is the first to go, then follow touch, muscle sense, and finally motor power. Return is in the reverse order, and a leg may be operated on long after movement has returned. If the injection is dilute, short analgesia is developed without loss of power. The size of a nerve root or trunk also determines the effect of a particular concentration of drug. Gravitational diffusion is an inaccurate method, wasteful of power, and uneven in distribution, for if the drug wanders beyond its intended area, loss of concentration results where it is most needed. But although moderate doses of novocain spread by gravity into the neck may not paralyse respiration, they undoubtedly impair the activity of the centre and the conductivity of the motor roots of the phrenics and intercostals in proportion to the concentration which each receives, the one through the blood and the others directly, for the respiration under high blocks is often reduced to a minimum.

Koster's Theory and Experiments.

There has lately been some attempt to reintroduce this method for operations on the head and neck, and the name of Koster, of America, has been prominent in this connection. Koster[6] exposed the medulla and upper cervical cord in frogs and guineapigs, and applied novocain solution direct to this region. He records that the animals became analgesic all over and that they also became somnolent. He also applied solution to mixed nerve trunks in order to demonstrate that sensory fibres are more easily affected than motor, a fact which was already known. He deduced from these experiments that the strength of novocain which was effective in interrupting the sensory conduction paths in this region did not affect the respiratory centre because this is a motor mechanism, and therefore not so easily affected. He also states that the somnolence was due to diffusion of novocain to the cortex. Applying these results to human beings he propounds the theory that all past fears of paralysing respiration have been groundless, and all that is necessary

is to keep the patient's head low in order to ensure a good blood supply to the medullary centres. All administration in the human being has, until recently, been by means of hyperbaric solutions. The anterior root block seen under hypobaric solutions of percaine revealed definite proof that the action of the analgesic is not direct into the cord but only on the roots, for it became evident that the motor side could be paralysed when the sensory fibres were left free. This disposes of the theory that muscular relaxation is due only to lack of stimulus, and suggests that the pia mater is impervious to analgesics; it should also be remembered that the spinal pia has a fibrous layer. Koster's experiments depend on the direct action of the drug through the pia mater into the conduction tracts. Before applying novocain he determined that the lumbar reflex arc was intact by stimulating the skin of the leg. After a presumed transverse section of the sensory conduction paths by novocain, he states that the animals became analgesic all over and that stimulation of the legs produced no response. Now 1 per cent novocain injected up the spine of human beings will paralyse motor fibres of the anterior roots, but Koster states that a $2\frac{1}{2}$ per cent solution applied to the medulla of these animals did not. Is it not obvious that this strength never reached the motor mechanism in this region? Did it reach the sensory tracts? Assuming that it did, what abolished the reflex arc in the lumbar region, for no novocain was applied here, and it could not have run down to the lumbar roots because the animals were in the Trendelenburg position? Was this due to central motor inhibition? Is it not possible that the animals were analgesic and somnolent from blood absorption? Whatever the explanation may be the experiments were based on a fallacy, and provide no material evidence whatever for a generalisation applicable in our operating theatres. One case of respiratory embarrassment on the operating table is worth a thousand such experiments.

Respiratory Embarrassment and Collapse.

The danger in intra-thecal administration is not a vaso-motor effect from a regional block of the thoracic and lumbar

roots, which proves to be of much less significance than was formerly thought, but from blood absorption and paralysis of the vaso-motor centre, leading in some cases with excessive dosage to severe collapse to a pulseless state. The nausea and vomiting which sometimes occur after injection, apart from surgical manipulation, is similarly due to stimulation of the vomiting centre by the poison, as well as low blood pressure. This may be realised by injecting the full contents of a spinal ampoule into a muscle. It is not to be expected, therefore, that the respiratory centre will escape entirely, and the degree to which it may be affected will depend on the amount of the dose in relation to the body weight of the patient. The effect on the cortex, shown by the drowsiness and tendency of the patient to sleep, also depends on the amount of the drug circulating in the blood. The zenith of blood content is probably reached about fifteen to twenty minutes after injection by the gravitational method, and a little earlier by the direct method, because of the larger absorptive surface to which the drug is immediately exposed.

Cushny[7] states that the relative non-toxicity of novocain is due to the fact that novocain is destroyed by the liver, and cocaine is not. The slow absorption from infiltrations, especially with adrenaline, gives the liver time to deal with the poison, but when novocain is injected intravenously the toxicity more nearly approaches that of cocaine. On account of the rapid absorption of spinal injections their toxicity must be reckoned as only less high than those administered intravenously. If the dosage is high and the body sloped, a variable concentration will reach the anterior roots of the phrenics and intercostals and reduce their conductivity in proportion to the concentration, and the respiration becomes shallow and slow. Anoxæmia will further reduce the centre and increase a tendency to sleep, for it is a powerful, and our last, anæsthetic.

The respiratory centre may therefore be paralysed by an overdose of the drug in the blood, or by a combination of reinforcing agents, of which the analgesic and a preliminary narcotic may be the chief. If the centre is badly fatigued and poisoned by drugs it may be difficult to keep it active,

whatever means may be adopted. An overdose of percaine, whether by infiltration or by intra-thecal injection, will paralyse the centre, and a competent hypobaric solution injected into the neck will paralyse the phrenics and intercostals. Koster states in relation to the human subjects, "In many patients, particularly in children, when head anæsthesia is induced by encouraging diffusion of the drug upward to the medulla, it is found that sleep results." This appears to have been the experience through the history of the method from the earliest cocaine days, and is but due to the fact that the dosage has been relatively high in children compared with body weight. This sleep has also been ascribed to the complete isolation of the consciousness from all body stimuli: A mind deprived of body sensation, yet capable of that most potent of all experiences, fear, and falling asleep on the operating table out of sheer boredom from want of stimuli? To produce a general analgesia, all that is necessary is to inject a sufficient dose of novocain in the lumbar region, thereafter inclining the body in the Trendelenburg position. This spreads the drug over the nerve roots, at the same time exposing it to an enormous absorptive surface. The general effect on the patient may be profound. Koster admits that in some cases a peripheral pulse is not to be expected, but implies that this is no disadvantage, since the surgeon can always refer to the aorta, a consolation not available to the specialist in anæsthesia. This condition almost resembles a state of suspended animation, and I have remarked that the patient is "hibernating." And so it seems. Every function of the body is depressed to its lowest level: no pulse can be felt or, when palpable, may be reduced in rate as low as thirty to forty per minute. The conductivity of the phrenics is reduced and respiration is just sufficient for life. A short period of sleep may be observed, and the lips and cheeks may puff out as they do under chloroform.

There are two components at work, a regional nerve root block and a blood dose of novocain. To what extent the cranial nerve roots are directly involved is an interesting question. In the dorsal decubitus the novocain will first be directed towards the cisterna magna, and from here it may

spread over the base of the brain, but the arachnoid reticulum is here at its densest and the amount of fluid far greater than it has already encountered in the spine. In such high dilution the novocain has to attack the largest and most compact sensory root in the body.

Is it not probable that an amount of novocain injected in the lumbar region, capable of spreading the sub-arachnoid space as far as the fifth cranial roots, may also be capable of producing general analgesia apart from nerve block, if not actually general anæsthesia?

Dickson Wright[8] is an enthusiastic advocate of spinal injections for operations on the head and thorax, but says that "shallow and slow respirations may be worrying." He records a respiratory rate of six per minute, and says, "The patient was asleep, and I did not care to wake her to tell her to breathe faster." When admonition is inapplicable, carbon dioxide and oxygen are indicated. The outstanding clinical features of such cases are a reduced frequency and amplitude of respiration, and very low blood pressure, sometimes reaching a pulseless state. The one means imperfect lung ventilation, and the other a reduced rate of blood flow through the lung capillaries. Both tend to anoxæmia and a vicious circle which may end in an arrest of respiration, and it is no recommendation to claim that the majority survive.

Jonnesco's Practice.

Jonnesco's practice was based on the fallacy of direct medullary action, and on the principle that strychnine injected with the drug stimulated the centre and kept it active. Strychnine has no action on nerve fibres, and to reach the cells of brain or cord must first be absorbed into the circulation. But cells paralysed by an analgesic would not respond to stimulants, nor would the latter avail to stimulate cells whose conducting fibres were blocked. Nothing but the possibility of damaging the cord prevents Jonnesco's puncture between D_1 and D_2 from being a practical method. Short analgesias in the area of the cervical nerves could be obtained by injecting small volumes of dilute solutions, and the motor roots of the phrenics

and some of the extraordinary muscles of respiration would have their conductivity reduced but not abolished. To gain longer analgesias stronger solutions would be required, which might paralyse the phrenics and some extraordinary muscles, leaving the intercostals to carry on respiration. But the rigid chests of old people would be a bar to such a procedure. It follows, therefore, that only short analgesias would be advisable by this method. Dilute solutions can be injected up from the lumbar region, but the large volume required means more drug for blood absorption. There must be a very dilute solution of percaine which could be operated in this manner, but what the strength is I cannot say at the moment. The 1–1,500 strength would paralyse the phrenics, and must not be injected into the neck.

Clearly separated from the foregoing is that kind of administration which is intended for abdominal and lower operations, the acknowledged territory of what is generally known as spinal analgesia. Such administrations have in the past frequently passed intended limits, and merged into general anæsthesia, from want of interpretation of the factors involved. The division comprises two subdivisions which require different treatment, limited thoracic nerve root block, and intra-dural caudal block.

Limited Thoracic Nerve Root Block.

Splanchnic block for abdominal surgery requires that the drug shall pass the roots of the fourth or fifth thoracic nerves. Between these and the fourth cervical roots is a sufficient interval to give a wide margin of safety. There are only two possible ways of spreading an analgesic over the roots from the lumbar region :—

1. Gravitational diffusion, which can be limited only by dosage and a moderate angle of the table, and gives an uneven distribution.

2. By manipulating a syringe :

(*a*) Using cerebro-spinal fluid; simple addition of drug is followed by gravitational diffusion.

(*b*) Direct injection of foreign fluid charged with the drug; if this is hyperbaric gravitational diffusion follows;

if it is isobaric or hypobaric no spread follows in the Trendelenburg position.

The adult spine is most variable in length. Measurements in my series of cases from the inter-iliac line to the seventh cervical spine, with the body in full flexion, have varied between sixteen and twenty-two inches, and both shorter and longer probably exist. In addition, the cubic capacity of the dura in women must be reckoned as rather less than that in men.

The Method of Barbotage or Replacement.

This is the method of withdrawing the cerebro-spinal fluid and adding the drug to it before reinjecting. This is impracticable with novocain, because a hyperbaric solution is always produced. Percaine may be used up to 3 c.c. of 1 in 200 dissolved in acidulated distilled water. This will produce an approximately isobaric solution, and will apply equal concentration to both anterior and posterior roots. Analgesia will therefore outlast abdominal muscular paralysis, but relaxation will be good. The amount of fluid withdrawn must be proportioned to the length of the spine, otherwise no constant nerve root level can be expected. The disadvantages of this method are that the withdrawal of a large quantity of fluid is tiresome and not always successful with a fine needle, and the addition of so small a quantity of acid fluid to a large quantity of alkaline fluid may result in the precipitation of the percaine. Quarella,[9] who favours this method, regards scopolamine and morphine as an essential part of the technique, and it may be that this is because the level of root block is not always what has been aimed at, for how can it be constant if the spine has not been measured?

The Method of Direct Injection.

This means the injecting of a dilute hypobaric solution of the drug without withdrawing cerebro-spinal fluid. This is the simplest method, a full account of which has been given elsewhere.[10] To calculate the volume of fluid required to pass the fifth thoracic roots, take a measurement from the inter-iliac line to the seventh cervical spine, with the body

in full flexion; from the number of inches recorded deduct four for men, and six for women; this result indicates the number of cubic centimetres of solution to inject between L1 and L2, or L2 and L3. This formula is put forward as a basis for a moderate increase with experience, and as a reminder that women require rather less than men. If the operation is pelvic the injection should be made between L4 and L5, in order to hit the lowest nerves in their most exposed situation. For gynæcological operations 10 to 12 c.c. should be ample.

In the dorsal decubitus a hypobaric solution tends to apply itself in greater concentration to the anterior roots, and in some cases may give abdominal muscular paralysis and no analgesia. In the lateral position, a unilateral effect is produced, a result of importance, because the upper affected side is at the disposal of the surgeon. The degree of this separation depends on the amount of the difference between the specific gravities of the two fluids, and the rate of injection. To produce this effect in the lateral position, make a slow injection, for the more rapid the injection the better the mixture of the two fluids. To ensure analgesia, the patient must be put in the ventral decubitus after injection from five to ten minutes; the duration of the analgesia will depend on the time spent in this position. If in any case this position seems inadvisable, the two lateral may be used consecutively. It will be found that as a result of the continued action of the drug on the anterior roots when the patient is put on the back that the paralysis of the abdominal muscles will probably outlast the analgesia. The nearest approach to an ideal drug for this method yet produced is percaine, because it is capable of producing a prolonged effect on nerve fibres in such high dilution that the rate of absorption into the blood is very slow. A rapid general toxic effect is thus avoided. It may be used in strengths of 1–1,000, 1–1,500, and 1–2,000, dissolved in acidulated 0.5 per cent saline (S.G. 1.0034). The normal saline solutions, S.G. 1.006, are midway between possible variations in the specific gravity of the cerebro-spinal fluid. By this method sufficient acid fluid is injected with the percaine to keep it in solution.

The latest fallacy to arise is that "percaine gives such marvellous relaxation." The perfect and lasting relaxation is due to the method of administration whereby the powerful drug is efficiently applied to the anterior roots. It is correct to say that isobaric and more especially hypobaric solutions give good relaxation, and that hyperbaric solutions spread by gravity tend to give poor relaxation in the upper segments of the abdominal wall. Thus a percaine glucose solution would have this defect. In spite of this profound regional root block the fall of blood pressure after percaine injections, though occasionally smart, is never so severe as after massive doses of novocain, nor is toxic vomiting and pallor so much in evidence. Novocain may be used in this way: 1 per cent dissolved in 0.25 per cent saline, S.G. 1.003 or in 0.5 per cent saline, S.G. 1.005. If 1 per cent in normal saline is used, S.G. 1.007, the patient may be kept horizontal in the dorsal decubitus until absorption has reduced the specific gravity, and both roots should be fairly evenly affected. The strength of these solutions may be increased in proportion to the faith of the user in the non-toxicity of this drug. When higher percentage is used sodium chloride must be left out proportionately to preserve the required specific gravity of the solution. The strength limit in which novocain can be used as a certain hypobaric solution is 2 per cent in distilled water. When dosage becomes high, blood absorption will dominate the picture.

Limited Intra-dural Caudal Block.

Intra-dural caudal block requires different treatment from that of thoracic root block, because the nerve trunks are more resistant than the roots. The 1 in 1,500 percaine solution when completely effective in the abdominal area sometimes leaves some power of movement in the legs, and deep sensation, showing that the lumbar nerves are more difficult to affect both in their roots and trunks than the thoracic. For limited caudal block stronger solutions are therefore required. The novocain and stovain solutions in common use always hit the cauda equina in their strongest concentration, and the most profound effect is in the legs

and perineum, but the only way they can be limited is by sitting the patient up after injection. Limited block in the horizontal position requires an isobaric or a hypobaric solution. Percaine dissolved in acidulated distilled water or 0.5 per cent saline may be used in strengths from 1 in 100 down, and the amount of drug need never exceed from five to ten milligrammes. Thus ½ to 1 c.c. of 1–100; 1 to 2 c.c. of 1–200; and so on. Six to eight hours' duration of analgesia is possible with this drug, but such prolonged effects are doubtful practice. A great variety of strengths may be used, the object being to effect a good local mixture with cerebro-spinal fluid at the site of puncture. When the operation is on the leg, the barbotage must be sufficient to spread the drug on to the first lumbar nerve, or higher according to the site of the operation. The duration of analgesia will depend on the amount of barbotage used and the dilution attained in the cerebro-spinal fluid. Novocain up to 4 per cent in distilled water may similarly be used.

General Considerations.

One of the most prevalent fallacies used to be that spinal analgesia is a method to be used when the patient is in too low a condition to take a general anæsthetic, and a common utterance was: "He is too bad for a general, better give him a spinal," and this in relation to serious cardiac disease. It hardly seems necessary to condemn the administration of massive doses of novocain, with the possible severe fall in blood pressure, when the mechanical efficiency of the heart and the state of the myocardium is defective. A profound fall in blood pressure means a reduced coronary circulation, and only a sound heart can be submitted to such an ordeal. It is certain that ephedrin cannot be relied upon to prevent fall of pressure, though it may alleviate it. It should be injected into a muscle or into a vein and not into the fibrous tissue of the inter-vertebral ligaments, where absorption must be slow. A limited caudal block or a block of a few thoracic roots may be undertaken when a more extensive effect would be inadvisable, and it is necessary to distinguish clearly between the risks involved in low and high operations, since the necessities of dosage and the vaso-motor

effects are greater in the latter. The advent of percaine undoubtedly extends the use of this method in poor risks, and the 1 in 2,000 solution will accomplish much in short abdominal interventions, with very low general toxicity. But this drug will not confer immortality on any one. High thoracic root block finds its best application in the muscular man about to undergo a gastric operation, who would otherwise require a harsh general anæsthesia to bring him into a good state of relaxation.

My own outlook is "This man is too strong for a general, better give him a spinal." For normal people spinal analgesia may be offered as an alternative to any one who wishes to avoid general anæsthesia, and its advantages in lung disease, especially tuberculosis and other pathological conditions, are well recognised. But because in bad risks spinal analgesia has disadvantages of its own, it does not always follow that its use is excluded in such cases, for the post-operative risk of general anæsthesia may be even worse. The temporary stimulation of ether may be the most certain way of getting a patient off the table alive, but the subsequent depression may yet be disastrous, and it cannot be expected that an anæsthetist shall devote the whole of his experience to avoiding the coroner's court, and qualifying his patient for a death certificate.

For such judgment as may be required in these cases no rule can be made. For operations on the head and neck this method cannot compare in regard to safety with a well-planned sophisticated gas anæsthesia by intubation methods, or a two-way insufflation of ether. In operations on the thorax when a mechanical difficulty in respiration already exists I believe no valid argument can be found in its favour, though a unilateral effect with 1 in 2,000 percaine may be of service for an operation on the lower chest wall. In relation to its employment for such operations as dissection of tonsils after injection of massive doses of novocain in the lumbar region, I am reminded of the criticism attributed to Dr. Johnson, when shown a little dog walking on its hind legs: "Sir, to be sure it is done, but it is not well done; the marvel is that it can be done at all."

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A SYSTEM OF ANÆSTHESIA FOR THE DENTAL ANÆSTHETIST.

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I MAINTAIN that by using nitrous oxide one can obtain the best possible anæsthesia from every point of view—the patient's, the surgeon's and the anæsthetist's, and obtain it in every type of case, however young or old, however diseased or healthy.

In order to give you an idea of my methods I must not only tell you what I do, but also try to give you my reasons for so doing, so that you can appreciate the difficulties that may arise during an administration, and my methods of overcoming them. Consequently I first propose briefly to give you an outline of the physiology of respiration, as it concerns the anæsthetist, then to give you a little physiopathology, leading up to the basic principle employed in nitrous oxide anæsthesia and, lastly, a few words on apparatus, followed by details of administration.

1. *Tidal air* is the quantity of air which is habitually and almost uniformly changed in each act of quiet, normal breathing. In a healthy adult man it averages about 500 c.c., which may be visualised as just under a pint (about 17½ oz.), and this at body temperature will be expanded to 600 c.c. (about 21 oz.), or just over a pint. This amount of air is, of course, not nearly enough to fill the lungs. Haldane gives the capacity of the upper air passages and bronchial tubes as 200 c.c. (7 oz., or the capacity of a teacup), and therefore about one-third of the tidal air is required to fill this dead space.

2. *Complemental air* is the quantity over and above this

tidal air which can be drawn into the lungs in the deepest inspiration. The average amount is about 1,600 c.c. (56oz., or about one-third of a gallon—just under three pints).

3. *Supplemental air* is that quantity of air which can be expelled from the lungs at the end of normal expiration, and is of about the same volume as the complemental air, i.e. 1,600 c.c.—or just under three pints.

4. *Residual air* is that which is left in the lungs after supplemental air has been expired. This, of course, varies, but it is roughly the same as both the foregoing.

5. *Respiratory or vital capacity* of the lungs is the greatest volume of air which can be expelled when the lungs are full of complemental air. In other words, the greatest quantity of air that can be exhaled after the deepest possible inspiration—or the sum total of complemental, tidal and supplemental air. In the average adult it is somewhere between 3,500 and 4,000 c.c. (between 6 and 6½ pints).

A well-trained deep breather could be made forcibly to expel all the tidal and supplemental air in his respiratory system, and inhale to the full extent of his vital capacity (about three-quarters of a gallon of gas). But in practice you would find the greatest difficulty in persuading anyone to do this; also, the sudden change would be unpleasant, and it should be remembered that the method one employs aims at being the pleasantest possible for everyone concerned, which this certainly would not be. In order to obtain a rapid induction it is not even necessary to make the patient breathe deeply; he will "go under" quite quickly enough if every inspiration consists of pure nitrous oxide.

From the foregoing it is obvious that if you have a three-gallon bag of gas, which is the normal size of a gas-bag, and you place it over the patient's face in such a way that he can only breathe in and out of it, these three gallons of gas will eventually be diluted with the air which is in his lungs to start with, i.e. the total volume of tidal, supplemental and residual air (about three-quarters of a gallon), plus the CO_2 and nitrogen which the nitrous oxide is driving from the blood. Thus it follows that the mixture he is finally re-breathing will be approximately 75 per cent nitrous oxide.

This was the method adopted originally when gas was first used for dental extractions, and it is not surprising that it produced little more than analgesia. In justification, it must be remembered that these pioneers had the excuse that gas was supplied to them in ox-bladders!

To-day we aim at more than analgesia. We aim at the production of a smooth and steady state of anæsthesia which can be prolonged indefinitely and controlled completely. In other words, we aim at the elimination of the nitrogen and other gases from the lungs, blood plasma and tissues of the patient, and their replacement by nitrous oxide.

If a normal patient be allowed to breathe a gas which does not supply him with oxygen he will become cyanosed, asphyxiated and anoxæmic.

Anoxæmia, cyanosis and asphyxia are not synonymous terms. Anoxæmia, for example, is not a modern word for asphyxia, as one writer naïvely defines it; it merely means an absence of oxygen from the blood. As anoxæmia cannot suddenly be produced, there will be various stages between normally oxygenated blood and anoxæmia, so we speak loosely of various degrees of anoxæmia. The name given to the train of symptoms caused by depriving a patient of oxygen is asphyxia.

With regard to cyanosis, it is possible to have a cyanosed patient who is not being asphyxiated, and vice versa. Cyanosis is merely due to there being more hæmoglobin than oxyhæmoglobin in the capillary circulation of the skin. Now, in the plethoric patient there is more hæmoglobin than is necessary; in other words, a small percentage of it can carry all the oxygen required for his physiological needs. This being so, the patient can easily be cyanosed without being asphyxiated. In an anæmic patient, on the other hand, there is so little hæmoglobin that all of it is required to carry oxygen in the form of oxyhæmoglobin, and the smallest diminution of the oxyhæmoglobin will produce asphyxia, but not cyanosis.

The signs of asphyxia under gas anæsthesia may roughly be divided into three stages: (1) the hyperpnœic; (2) the heteropnœic; (3) the apnœic.

In the first, or hyperpnœic stage the organism, feeling a

desire for more oxygen, first tries to breathe in as much as possible, with the following result:—

(*a*) The respiratory rate is increased. Expiration is unchanged, but inspiration is deeper—it is exaggerated, the patient takes longer to fill his lungs. This exaggerated inspiration is brought about by calling into action all the accessory muscles of inspiration in the following order:—the laryngeal, the small muscles of the face (e.g., the alæ nasi), the pectorales and trapezii, serrati magni, and the sternomastoids.

If the supply of oxygen be not forthcoming as the result of these tactics, they are reversed. Instead of trying to breathe in more, an attempt is made to breathe out more, in order to try to expel this useless, unsatisfying gas, with the following results:—

(*b*) Inspiration returns to normal, but there is an increase in the expiratory efforts, which become excessivley prolonged, being aided by all the accessory muscles of expiration, such as the abdominal, the internal intercostals, the triangularis sterni, the serrati postici inferiores and quadrati lumborum. This stage merges into the second, or heteropnœic stage. The expiratory efforts now become excessive and, rapidly giving way to general convulsions, cause the condition known as jactitation. This stage is very short, and the patient relapses into the third, or apnœic stage. Here, respirations almost cease, the pupils dilate, and the spasms give way to rigidity of all the accessory muscles of respiration, in which, of course, are included those of the anterior abdominal wall, causing opisthotonos, pleurothotonos, etc., according to which set of muscles is the stronger. If the state of anoxæmia is not relieved the respirations become few and far between and are sighing in character, until they cease altogether. The muscles relax; the pupils by this time will have contracted somewhat, the heart will be beating slowly and fully, and. sometime after respirations have stopped, it also will stop.

Having, I hope, made it clear that anoxæmia, cyanosis, and asphyxia are not synonymous terms for the same condition I will now attempt to describe how I produce a state of anæsthesia with gas.

The basic principle employed in anæsthetising a patient

with gas is the replacement of the nitrogen and oxygen in his tissues as well as in his blood by nitrous oxide and oxygen in proportions controlled by the anæsthetist.

I have been looking at the chapters on nitrous oxide in most of the books on anæsthesia that have appeared since the war, and find that most of them divide the subject into three groups—gas alone, gas and air, and gas and oxygen—each, apparently, being considered to have different signs of anæsthesia and different results.

This, of course, is not the case. The signs of gas anæsthesia are the same whether pure gas is used or whether it is diluted with oxygen obtained from the air or from a cylinder. The symptoms which these books describe as signs of gas anæsthesia, which they distinguish from those of gas and air, and gas and oxygen, are neither more nor less than the signs of asphyxia on the top of those of anæsthesia.

It is true that the dental student, for examination purposes, is expected to know how to perform this "beneficial asphyxia," as someone once called it, without doing the patient any permanent harm, so there is some justification in giving instructions how to do this. But it should be remembered that the dental student is not being trained as an anæsthetist; all he is expected to be able to do is to render the patient anæsthetic as safely as he knows how, should the necessity arise.

Just as the signs of ether or chloroform anæsthesia are the same whether one uses oxygen from the air or from a cylinder, so are they the same with nitrous oxide, whether air or a cylinder be the source of the necessary oxygen.

In the majority of cases it is not possible to get a smooth anæsthesia until the nitrogen has been displaced from the tissues and its place taken by nitrous oxide. Nitrous oxide is far more soluble in blood plasma than nitrogen—100 c.c. of blood at body temperature will dissolve at atmospheric pressure about 50 c.c. of nitrous oxide, as against 1 c.c. of nitrogen—and, for reasons we learned in the physiology laboratory, nitrous oxide alone will displace the nitrogen from the blood more quickly than when mixed with oxygen. It is for this reason that I always begin with pure nitrous oxide, and do not at first allow rebreathing, thus ensuring

that there shall be a minimal dilution of the gas in the respiratory passages. Unfortunately, by using gas alone, the oxygen in the blood is also displaced; but anoxæmia of short duration—anything up to a minute—is harmless so long as it is relieved at the end of that time. The cyanosis so produced is incidental to a rapid displacement of the nitrogen from the blood. Once having established anæsthesia there will be room for sufficient oxygen to keep the average patient oxygenated.

Before I continue, it might interest you to know that, in 1928, when I was demonstrating my methods in Hamburg, much alarm was caused, at first, by my giving the patient pure nitrous oxide without oxygen. The reason put forward for this alarm was that one of the regulations of the hospital laid down that gas should never be administered without at least 10 per cent of oxygen mixed with it. I was also given to understand that this rule was not unusual in other German hospitals.

To return to our basic principle. In order to change the gaseous content of the tissues, which must be done via the blood, it is first necessary to change that of the lungs. This is done by allowing the patient to breathe in pure nitrous oxide only at each inspiration. At the end of the first inspiration only the tidal air will be replaced, some of which will diffuse into the reserve air. When you remember that the volume of the tidal air is about 600 c.c., the reserve air about 1,600 c.c., and the residual air another 1,600 c.c., you will realise how useless it is to give gas diluted with oxygen from the beginning, as you merely delay the onset of anæsthesia. I hope later on to show you that this is also harmful from the anæsthetic point of view.

Having displaced the nitrogen from the blood the resultant anoxæmia is tempered and controlled by means of oxygen, administered in exactly the quantity desired by the anæsthesist through the agency of one of the modern, finely adjusted instruments which we now have at our disposal.

With this basic principle before us there is no need to stress the fact that unsuitable apparatus and unskilled administration render nitrous oxide a dangerous anæsthetic agent: but with the requisite clinical experience, a cool head,

and one of the modern gas and air, or gas and oxygen machines the bogey of nitrous oxide anæsthesia—cyanosis and asphyxia—can be laid to rest.

Now a few words regarding instruments. These can be divided into two great classes: those where the gas flows continuously from the cylinder at a rate controlled by the anæsthetist, and those where the flow is intermittent, being controlled by the patient's respirations. They both have their adherents. Personally, I use both types, being guided in my choice by the kind of anæsthetic I propose to give.

It is very difficult to give a smooth nitrous oxide anæsthesia in every case with the continuous flow type of apparatus, unless one uses that trace of ether of which the adherents of this machine only sometimes grudgingly admit the necessity. With this trace of ether it is comparatively easy to give a good anæsthetic. I favour this type of instrument for abdominal work where a quiet relaxed abdominal wall is required, and where I frankly give ether vapour as an adjuvant—in small quantities, certainly, but ether, nevertheless.

With the intermittent flow type of machine, on the other hand, it is possible to use nitrous oxide, and nitrous oxide only, as an anæsthetic in 100 per cent of cases with good results. This is due to the fact that the gas does not flow until, and unless, the patient inhales, when it flows in just the quantity the patient requires at the pressure the anæthetist desires. Consequently no rebreathing is necessary, as is the case with the other type of machine. Also, as there is no rebreathing, the percentage of oxygen in the mixture can be varied from breath to breath: to take an extreme case, it is possible in the intermittent flow machines to give one breath of pure nitrous oxide and the next of pure oxygen.

The commonest continuous flow instrument in this country is Dr. Boyle's, which is a modification of the original Gwathmey instrument. There are numerous modifications of this instrument, all designed toward making the control of the gases easier. They all use the same principle of the continuous flow of the gases through various holes submerged in water. There are other continuous flow machines, such as the Heidbrink, which make use of pressure gauges to indicate

the flow of the gases. All these instruments necessitate some degree of rebreathing.

The best known of the intermittent flow machines is the McKesson. Anæsthetists all over the world have to thank the mechanical genius of Dr. McKesson for producing a machine which enables them to do with gas and oxygen exactly what they want, when they want, merely by the movement of one lever. The only other adjustment required is the manipulation of the pressure screw in order to vary the pressure of delivery. Dr. McKesson has recently perfected a great improvement on his original machine, the main feature of which, to my mind, is the controlled rebreathing, controlled not only as to amount (which, of course, was a feature of the old machine) but controlled also as to pressure. There is a spring on the rebreathing bellows, the tension of which can be varied at will, so enabling rebreathing to be used much more than was formerly the case.

There are only two methods of administration with which the dental anæsthetist need be familiar. The first is the nasal administration of nitrous oxide, with oxygen either from the air or from a cylinder, and the second is the nasal endotracheal administration of the gas with oxygen from a cylinder. This brings us to the question as to whether we should use oxygen from the air or from a cylinder, a question which is not quite so simple to decide as some maintain. It is true that the administration of oxygen from the air has one great advantage over oxygen from a cylinder—the apparatus required for it is simple and cheap, light in weight and easily portable, whereas that required for oxygen from a cylinder is delicate to handle and expensive, and has the added disadvantage of being weighty and cumbersome. On the other hand, the maintenance of smooth anæsthesia with gas and air is difficult, and can only be attained after much clinical experience, particularly in the case of children, while the technique of gas and oxygen is, by comparison, simplicity itself when a modern instrument is used: very little clinical experience is needed to attain proficiency, and children can be anæsthetised with comparative ease and safety. Another reason why gas and oxygen is preferable to gas and air is that with the former, once having washed all the nitrogen

out of the system and replaced it with nitrous oxide and oxygen, it is possible to give more oxygen than when there it still nitrogen present in the tissues.

One curious fact concerning gas and air is that it is a better analgesic than gas and oxygen. The late S. R. Wilson demonstrated this during the last paper read by him at the Royal Society of Medicine.

The method I prefer is to administer gas and air for short anæsthesias of about five minutes duration, and gas and oxygen for longer ones.

There are two ways of anæsthetising a patient with gas—the slow and the rapid. From every point of view I consider the rapid to be the best. It is kinder to the patient because it shortens the trying pre-anæsthetic period for him. It is better for him physiologically, because a rapid induction means that he is having the minimum of gas and, therefore, after a short operation he will recover more quickly; and, thirdly, the psychic factor of shock is eliminated because the excitement stage is avoided, for excitement in the early stages of anæsthesia throws an unnecessary strain on a heart already embarrassed by a poison. In this context it is well to remember that the frame of mind of the average patient, even before so simple an operation as a single extraction, is that of suppressed excitement and apprehension. If the gas be given well diluted with air and oxygen the first few breaths of it only accentuates this, with the result that he starts to worry, and to imagine that all manner of things are happening, or are about to happen, to him; so he tries to stop the further administration of the anæsthetic, which he realises will eventually overcome him. Hence the struggling we so frequently hear about during slow induction, which, in the case of a diseased heart, may be the cause of death before the operation is even begun. The only way out of this difficulty is to get the patient "under" before he has time to think.

The Management of Out-patients Operated upon in the Chair.

In the first place I always endeavour to meet my patient's wishes as to his comfort. When he has settled himself down in the chair it can easily be tilted to suit one's convenience.

The old method of making him sit bolt upright with legs and thighs at right angles and the head vertical is not only unnecessary but is most uncomfortable. I do not use a prop on a nervous patient unless it be absolutely necessary, but if one should be used I place it as far back as possible and start giving the anæsthetic immediately it is in position.

It should not be forgotton that gas is an anæsthetic at normal temperature and pressure, and this being so it should never be forced upon the patient. He should literally be wafted into a state of anæsthesia with the gas blowing gently from the nose-piece over his nose and mouth. If he should suddenly begin to breathe through his mouth after having been breathing through his nose it is either a sign that it is not being made easier for him to breathe through the nose than through the mouth, or, alternatively, that he is receiving insufficient oxygen. When you are out of training and you run for a bus you begin to breathe through your mouth, although until then you have been breathing through your nose. The same thing happens when a patient feels a desire for more oxygen during gas anæsthesia.

Whether I am using gas and air, or gas and oxygen, I always begin with pure nitrous oxide without rebreathing for reasons that have already been detailed. It is not the presence or absence of air or oxygen in the gas which makes the patient feel suffocated, but the mere fact of having something placed over his mouth and nose. If you doubt this, hold an ordinary face-piece with valves attached but without the bag on your own face, for, say, half-a-dozen breaths. Although you will be breathing air you will feel a sense of oppression, if not of actual suffocation, and the longer the face-piece remains on the more this feeling will increase.

I have the gas blowing out of the nose-piece before I approach the patient, and I let it blow over the nose and mouth for several breaths, lowering it the whole time gradually, until, by the fifth breath, the nose-piece is in position.

As it is important to make it as easy as possible for the patient to breathe through his nose, if he should appear to be having trouble while conscious, e.g., making audible sniffs, it is advisable to assure oneself that the sides of his nostrils are not being pressed upon, and then, if that be not the case,

to increase the pressure of the delivery until he inspires easily. When the pressure of the gas in the bag is increased, it is necessary to increase the pressure of the spring on the expiratory valve, otherwise the gas merely blows off and the patient derives little or no benefit. Pressure, in the ordinary sense of the term, must not be used on a conscious or semi-conscious patient or there will be a voluntary inhibition of respiration. This is a protopathic reflex designed to prevent emphysema of the lungs, and like all the other protopathic reflexes one of the last to disappear. All those who have walked against a gale will remember how difficult it is to breathe against the full force of the wind, and how they instinctively turn their heads to minimise this force. The patient experiences exactly the same difficulty when the gas is administered under pressure greater than is required to make it easy for him to breathe. The general rule regarding pressure is to use the minimum necessary to enable the patient to breathe easily in and out of the nose-piece. The breathing, of course, must be entirely through the nose; never in by the nose and out through the mouth.

The mouth-pad should be used merely as an obturator when necessary, which is not often. It can be used as such whilst adjusting the pressure of gas to make it easier for the patient to breathe through the nose than through the mouth. The only time to use it as a means of giving gas is when the nose is completely blocked; and in these cases it is really better to use some other form of anæsthesia, such as endotracheal.

As soon as I have produced a surgical anæsthesia, by which I mean a steady, quiet state of anæsthesia—and the only sure and reliable sign of this condition is the breathing, which becomes automatic, like the breathing of one asleep—I slowly increase the oxygen until the patient becomes normal in colour. If air is being used remember that it cannot be given continuously with nitrous oxide. The nitrogen that is admitted with the oxygen not only dilutes the nitrous oxide but also the oxygen, with the result that, if air is given in definite proportions with nitrous oxide, the patient if he is getting sufficient oxygen will not be getting enough nitrous oxide to produce anæsthesia, or, alternatively, if he is getting

sufficient nitrous oxide to produce anæsthesia he will not be getting sufficient oxygen to supply all his needs. This is the cause of the cyanosed, screaming patient sometimes met with by the inexperienced administrator. If air is to be the source of oxygen it must be given intermittently. As the patient is finishing one expiration the gas is prevented from leaving the bag, so that the next inhalation consists of air. The succeeding exhalation is dispersed into the air, and at the end of this the stop-cock is opened and the patient breathes nitrous oxide again. The whole success of a nasal administration with gas and air depends on the way the air is admitted, and on keeping the nitrous oxide free from dilution with air. If a gas and oxygen machine is being used the lever is turned to 10 per cent, this being a good average to go straight on to, and varied as necessary until the patient is "steady," but no rebreathing of any kind is permitted.

It is just as important not to give too much oxygen as not to give too little, but the signs of whether the patient is having too much or too little are not so simple to interpret as some people would try to make out. Should he become restless this condition may be due either to the presence of either too much or too little oxygen. Clinical experience is the only guide in determining this. If gas and oxygen is being used, and too much oxygen is given so that the patient starts to make voluntary movements, a few breaths, administered immediately, of 100 per cent nitrous oxide will overcome this restlessness, and a return can then be made to less oxygen than the percentage which caused the trouble.

If a patient should become light enough to feel what is going on, even though it does not hurt him, he may attempt to let you know that he is conscious, with the result that he may become restless or scream, or both. Once a patient has done this it is as difficult to get a quiet, steady anæsthesia again as it is to get this in a nervy or apprehensive patient from the beginning. It may be compared to forcing a nut on to a bolt with the threads crossed, which can be done with difficulty, but is not so satisfactory as when the nut is on the correct thread. All those who have had any hospital experience will have noticed that when once a patient has made a noise in the gas room the three or four succeeding patients are

more than ordinarily difficult to get "steady." The mental attitude of the patient plays a far greater part in contributing to smooth anæsthesia than most dentists would appear to realise, which is cogent reason for the dentist himself cultivating a calm and confident demeanour in the handling of his patients.

With gas and air the extraction can begin as soon as the patient is "under"; with gas and oxygen I like to find the patient's right mixture before the operation starts.

I always place gauze or gamgee in the mouth to act as an obturator to the pharynx. If this is done properly in every case there can be no trouble arising from foreign bodies entering the lungs during extractions. Besides the above reason, the use of gamgee or gauze enables the tongue to be controlled; and, lastly, the patient can be allowed to recover slowly with his head back on the rest without fear of his swallowing blood. To pack the mouth I place gauze or gamgee under the tongue, and fold the tongue back out of the way. An alternative method is to pack gauze or cotton-wool behind the upper centrals, and to keep on packing between the gauze already there and the teeth so as to form a bolus on the back of the tongue without pressing on the soft palate.

Cigarette fiends, feeble-minded and other abnormal patients all present difficulties which have to be treated symptomatically. I find that drunkards and drug takers are easier to manage if they are told to take a double dose of their usual dope about an hour beforehand. This is contrary to the usual teaching, but it is none the less true. They are, however, especially sensitive to oxygen starvation, so one has to be most careful not to let them feel suffocated whilst conscious, more especially semi-conscious, or insufficiently supplied with oxygen when anæsthetised, otherwise they become unmanageable.

The Management of In-patients done on the Operating Table.

For cases done on the table, when they are straight ones, such as straightforward, easy clearances, or operations on teeth other than molars, I use a nasal—preferably gas and oxygen. If the operation is on the molars, especially on the

third molar, or if it is a cyst, or any case where there is likely to be much blood or pus in the mouth, I use the endothracheal method. A point I wish to stress is that one should never do more to a patient than the circumstances call for. It is not necessary to make a routine of doing endotracheal administration on every table case, in fact I consider this is a bad practice. This method should be reserved for long cases where there will be much blood or fluid in the mouth, or where the surgeon has a delicate operation to perform in which he requires a perfectly still patient, and the anæsthetist well out of the way. In wholesale extractions, or any table case where oral packing will not inconvenience the surgeon, nasal gas and oxygen can be administered with advantage. If the mouth is properly packed—personally I prefer gamgee for this purpose—it is quite easy to keep the pharynx free from blood.

Preparation of In-patients for Operation.

Table cases are easier to do than cases in the chair, because one can give any premedication that one wishes. One of the main reasons for using gas and oxygen, as opposed to other anæsthetic agents, is that it enables sufficiently large doses of premedication to be given without having to worry over respiratory depression. My ideal is to have the patient unconscious before he leaves his bed. He should be placed on the canvas of the stretcher before receiving his premedication, so that, when the time comes to take him to the theatre, the poles have only to be inserted and he can be lifted on to the trolley, and from the trolley to the table, with as little risk of disturbing him as possible.

The routine injection I employ is omnopon gr. 2/3 (0.040 gm.), scopolamine gr. 1/150 (0.0004 gm.) given an hour before operation. If I want to make certain that the patient will be asleep before being taken to the theatre I give avertin in addition, from 50 to 75 mg. per kilo, at the same time. If, for one reason or another, avertin cannot be given, and the patient is wide awake when I arrive, I give an intravenous injection of omnopon gr. 1/3 (0.020 gm.), or. more recently, nembutal, either orally or intravenously.

I should like to say a word here about the use of atropine

in premedication. This is one of the worst drugs to give before gas and oxygen, especially by itself. A patient who has been given atropine, gr. 1/100, is most difficult to anæsthetise with gas and oxygen—more so than if he had received no premedication of any kind. I shall not attempt to theorise here upon why this should be so, but it is the case. It will be found also that when atropine is combined with any of the derivatives of opium it detracts from their beneficial effects. It is much easier to give gas and oxygen to a patient who has had morphia, gr. 1/3, without atropine than when atropine has been added. The same remarks apply to morphia used with hyoscine, these being more effective without than with atropine.

I first realised this at St. Bartholomew's Hospital, where we were accustomed to use a mixture known as Hyoscine A Compound or Hyoscine B Compound, consisting of morphia, hyoscine, and atropine, before the administration of gas and oxygen. The difference between the A and B compound is one of dosage. I found that morphia with hyoscine was far more effective alone than when combined with atropine. Hyoscine is quite as effective for preventing salivation as atropine, and it is certainly more sedative. I also found that atropine was not only useless when combined with morphia and hyoscine, but that it was positively harmful from the anæsthetic point of view. Whilst experimenting with various drugs I came to the conclusion that by far the most effective from the gas and oxygen point of view was a combination of omnopon and scopolamine, of which I found the average adult dose to be omnopon gr. 2/3 and scopolamine gr. 1/150. This I now use as my standard, and I give it to all ages from about eight upwards, in doses of from one-quarter of the standard dose upwards. For patients over sixty and under sixteen omnopon and scopolamine should be given in smaller doses, usually from one-half to two-thirds of the standard.

Morphia and hyoscine are not nearly so effective as omnopon and scopolamine, which latter is put up in ampoules in this dosage, as well as in two other doses by the makers. Omnopon, which is a proprietary extract of opium, known as pantopon on the Continent, is better for the patient from the anæsthetic point of view than morphia, and scopolamine is

the lævo-rotatory variety of hyoscine, which is far more sedative than the other two varieties. It has been stated that the hysterical results which are sometimes seen after hyoscine, but not after scopolamine, are due to an excess of dextro-rotatory variety in the former.

I use omnopon and scopolamine even though I am giving avertin rectally or any of these new intravenous barbiturates. If avertin is administered after omnopon and scopolamine, it is only necessary to use from 50 to 75 m.g. per kilo to render the patient somnolent.

In very nervous patients I find that gr. 3 of nembutal, given by mouth an hour before operation, followed by the omnopon and scopolamine injection a quarter of an hour later, is very effective. For children, nembutal is useful given either orally or rectally, together with elixir of chloretone and liquid extract of belladonna (which is better than either the tincture or liquor atropini) given by mouth. For children, however, I prefer to use avertin.

Exceptions do arise when one is suddenly called to a case in the country and the necessary drugs are left behind. Then morphia and atropine must be used. In this case I should use morphia in large doses (gr. 1/3) and atropine in small ones (gr. 1/200 to 1/150), and for infants and children from gr. 1/400 to 1/200. Children may tolerate atropine better than adults, but its use will make anæsthesia more difficult. A small trace of ether vapour, however, added to the gas and oxygen will get over this difficulty.

Should the patient's respiratory rate drop below nine to the minute, an intramuscular injection of 10 m.g. of alpha-lobeline will restore it to normal. Alpha-lobeline, known as lobeline in this country, is the only drug that is of any use in anæsthetic emergencies. Most of the others, such as pituitrin and strychnine, are worse than useless. Adrenalin certainly has a great use, but it is only secondary to lobeline. I always carry alpha-lobeline in 10 m.g. doses. It is the only efficient respiratory stimulant we possess. Sooner or later, if you use derivatives of opium and these new barbiturates, you are going to want it and want it badly. It is not likely to be needed so much during administration as after. A very likely time to need it is when you see the patient just before you

leave. He may be cyanosed and breathing very shallowly: in such a case an intramuscular injection of 10 m.g. of lobeline will work wonders. In emergencies it is given slowly intravenously—preferably with 50 gm. of glucose in 10 per cent solution.

It is most important, in order to get the full effect of the omnopon and scopolamine, to plug the patient's ears with vaselined cotton-wool, and to draw the blinds. In hospital a towel is put over the eyes. One should aim at a somnolent if not actually a sleeping patient arriving on the table for the anæsthetic. This brings me to another point—the less a patient is moved under an anæsthetic the better for that patient; therefore anæsthetise him on the operating table wherever possible.

Now for the details of a nasal endotracheal gas and oxygen. As soon as the patient is on the table I insert a small dental prop between his teeth on the left side—if there is sufficient room between the teeth to insert a gag on the right there is no need for the prop. Immediately the prop is in position the administration is begun with 100 per cent nitrous oxide, the pressure screw being turned to make the gas flow continuously. The face-piece is then gradually lowered until the expiratory valve can be heard working and the flow becomes intermittent. By this time the breathing should be automatic. The dial is then moved to 10 per cent oxygen for a few breaths and back to 100 per cent nitrous oxide until I estimate that the patient has received enough gas to keep him under for about a minute.

The mask is then removed, the right nostril lubricated with lubafax, Dennis Brown's or Waugh's gag inserted on the right side of the mouth, the prop removed and the teeth and lip protector placed over the upper left teeth and lip. The nasal tube is inserted through the lubricated nostril and visually picked up in the naso-pharynx with Magill's direct vision laryngoscope; the epiglottis is lifted up by the same means, and the tube is introduced through the cords, if they be separated. If they are not, the tip of the tube should be allowed to rest just against them so that when they are moved apart, either by the next breath or by a cough, it can quickly be pushed through them. The tube is of soft rubber, and

is controlled by the right hand manipulating the end outside the nostril.

It is better to use lubafax than vaseline, as the latter has a solvent action on rubber. It is also better to lubricate the nostril rather than the tube, as a dry tube is more easily managed than a greasy one. Dennis Brown's and Waugh's gags are both self-retaining; they are designed for use on the molars, and having a broad surface cannot damage the teeth.

The teeth and lip protector consists of rubber-covered sheet lead, as used by those who work with radium. After the gag is inserted the sheet-lead is bent over the left upper teeth and lip from laterals to premolars. In this way the upper lip is protected from being pinched between the direct vision laryngoscope and the teeth and the latter also are protected from being chipped or damaged.

The nasal tube should be of a soft rubber tubing which has been kept long enough in a coil to have a natural curve, and it should be the largest size that the nostril will take. Unless the tube be soft, some bleeding will be caused by its insertion, and even a soft tube may cause bleeding where there are adenoids or polypi.

Magill was the first to put a battery in the handle of the direct vision laryngoscope, so doing away with those wires which were the bane of our existence.

The tube is connected to the breathing tube of the machine by an angle piece, fitted with an expiratory valve, so that the patient breathes in and out through the valve.

I have purposely not mentioned the position of the head until now, as, generally speaking, the position in which the patient has been anæsthetised will be the correct one—head on the pillow, chin pointing to the ceiling. In some cases if the pillow be just pushed under the shoulder all necessary movement will be obtained. Usually, the easiest position is with the head on the pillow.

As soon as the tube is in place, with about a yard of soft, four-fold, nine-inch gauze, moistened with liquid paraffin, I start packing the pharynx around the tube, pushing the gauze well down with my finger. About half or more of the gauze is used in doing this, and the naso-pharynx is then

loosely packed with the rest. It is most important not to forget the naso-pharynx, as it is difficult to remove blood or pus therefrom after the operation. All this will have taken less than five minutes to do from the first application of the face-piece, and the patient can then be handed over to the surgeon with the confidence that nothing he can do in the mouth will obstruct the airway.

Summary:—

1. Nitrous oxide, properly administered, is the safest anæsthetic agent for all ages and conditions.

2. It is ideal for all operations on the mouth and pharynx.

3. It is best administered undiluted until the patient is anæsthetised.

4. Asphyxial symptoms should never be allowed to persist, but should immediately be relieved by oxygen—either from the air or from a cylinder.

5. It can easily be administered by the nose without interfering with the surgeon.

6. For extensive surgical work on the mouth it is best administered via a tube passed through the nose into the trachea.

7. Every opportunity should be taken to make use of premedication—especially in children. There is no need for any child to know anything about anæsthetics or operations.

PREMEDICATION.*

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FOR the purpose of this discussion, by *premedication* is understood a new conception of pre-anæsthetic medication, whereby the patient is rendered unconscious in his bed before the administration of the anæsthetic. The drugs employed for this purpose and the technique of their administration it is the object of this meeting to consider. The method has been aptly named *basal narcosis.*

It is no longer necessary that a man about to undergo an operation shall first of all be subjected to the ordeal of being taken from his bed to the theatre, compelled to witness the preparations for his operation, and then undergo the unpleasant experience of inhaling the anæsthetic until the last threads of his dissociating consciousness finally break. Neither is it necessary that when he once more awakens he shall reek of the anæsthetic, nor be distressed by hours of vomiting.

History.

In 1913 Gwathmey introduced a method for the production of narcosis by rectal oil-ether solution; and, although this method had certain disadvantages, it marked a great step forward as far as the patient's mental reactions were concerned. I have always felt that sufficient due has not been given to Gwathmey for recognising what a tremendously important thing is the abolishing of pre-operative fear. Some years later he published a paper on synergistic anæs-

* Read in opening a discussion in the Section of Anæsthetics at the Annual Meeting of the British Medical Association, Eastbourne, 1931.

thesia which marked the actual birth of basal narcosis as it is known to-day. It was this paper which stimulated me to try several drugs with the object of finding a method whereby a patient might safely be put to sleep while still in bed, and thereby saved the mental distress which often has such a deleterious effect upon his condition before, during, and after operation. I finally decided upon paraldehyde, which had been used by Gwathmey in his oil-ether mixtures. In spite of its disadvantages of smell, and in face of a good deal of opposition, the use of rectal paraldehyde steadily increased for some years.

In 1926 avertin was introduced by Professor Eichholtz. It is given in 2½ per cent solution per rectum, and is a much more powerful drug than paraldehyde. It lacks the disagreeable smell of the latter and has the additional advantage that it is only necessary to inject a small volume of the solution into the rectum.

Towards the latter end of 1929 barbituric acid and its compounds began to receive consideration both as anæsthetics *per se* and as complementary or basal narcotics. The barbiturates were well established as narcotics in general medicine. In 1924 Fredet and Perlis reported in the *Presse Medicale* the intravenous use of somnifeine as an anæsthetic. I tried it and was much impressed by the results; but reports that its originators were not altogether satisfied with it, and the fact that some dogs which were anæsthetised for research work also died, caused me to abandon it. I have also tried luminal given by the mouth, in doses of half to three-quarters of a grain per stone weight, and although complete unconsciousness was not obtained, patients were rendered drowsy and required less anæsthetic. It is to American workers that we owe the introduction of the newer, quickly acting, barbiturate compounds amytal and nembutal. Pernocton, of German origin, is very like the latter. Without being unduly enthusiastic, I think one may say that these compounds (and especially nembutal) have already proved of great value as basal narcotics; and I feel certain that they will be widely used in the future. They were introduced into this country by Magill, who brought supplies of amytal and nembutal back with him after his

visit to America with the British Medical Association last year, and it is to him that I owe my introduction to these drugs.

I propose briefly to consider each group of drugs, mentioning their characteristics and giving typical illustrative cases from my own experience; detailing also cases in which I have seen adverse symptoms. I shall purposely refrain from expressing any opinion as to the comparative value of the different drugs until the end, so that the descriptions may be as unprejudiced as possible.

Paraldehyde.

Except in young children and very old and debilitated subjects, paraldehyde alone will not produce unconsciousness. The addition of morphine or some other such drug is needed. Patients are graded into four groups. (This classification may also be of help in deciding the dose of other narcotic drugs.)

Group 1—Children under 7. Debilitated or severely toxic subjects. Patients with a high temperature.

These cases receive 1 drachm of paraldehyde per stone weight three-quarters of an hour before operation. The solubility of paraldehyde in water is 1 in 10, so that every dram of paraldehyde is dissolved in 10 drams of warm saline. This is shaken hard in a bottle to ensure solution, and run slowly into the rectum by catheter and funnel; the rectum having been cleared by saline enema at least two hours previously. If the patient becomes unconscious during the introduction of the solution further administration is stopped; but this only applies to Group 1. Atropine is given hypodermically where an anæsthetic other than gas and oxygen is to follow.

Group 2.—Normal adults and children over 7. One and a quarter hours before operation the patient receives hypodermically morphine gr. 1/40 for every stone weight, followed in fifteen minutes by rectal paraldehyde, 1 dram per stone weight.

Group 3.—Alcoholics. Athletes over 25 years of age. Very nervous patients. These patients receive on the night

before operation (repeated during the morning, if the operation is in the afternon) a full dose of bromide and chloral or bromidia. One and a quarter hours before operation: morphine, gr. 1/40 per stone weight, combined with hyoscine hydrobromide, gr. 1/150–1/100 hypodermically. One hour before operation: paraldehyde, 1 dram per stone weight.

Group 4.—Thyrotoxic cases. A preliminary trial of the patient's reaction to hyoscine should be made some days previously, as it sometimes causes great excitement. Preliminary sedatives—bromide and chloral, and morphine and hyoscine (if not contra-indicated)—are given as for Group 3. Owing, however, to the fact that paraldehyde may not be sufficient to produce sleep, it may be necessary to follow on with ether. The paraldehyde is therefore dissolved in olive oil—half an ounce of oil to every dram of paraldehyde—and if the patient is still awake half an hour before operation, one-half to two ounces of a 50 per cent mixture of ether and olive oil is run into the rectum, in half-ounce doses, at intervals of five minutes until he falls asleep. This is not, of course, an unvariable dosage: it is necessary to sum up a case, and a certain amount of experience is necessary. Most patients retain the rectal injection without trouble, but a suppository of chlorotone gr.*x* is of help in this respect. The average case is asleep in from twenty to twenty-five minutes, but some take as long as forty-five minutes; the rate of absorption from the rectum varies.

Illustrative Cases.

Male aged 48.—Operation for double radical antrum. Heart, second pulmonary sound accentuated, nil else abnormal. Blood pressure 140/90. Chest, hyperresonant, emphysema. Weight 12 st. 8 lb. Plethoric, drinks fair amount; big smoker; muscular; fat. Night before operation: bromidia 2½ drams. 7.45 a.m., morphine gr. ⅓, hyoscine gr. 1/100, and atropine gr. 1/100. 8.0 a.m., paraldehyde 12 drams in saline 15 ounces per rectum. 8.30 a.m., asleep; blood pressure 130/90. 9.0 a.m., anæsthetised, tracheal tube passed with ease using only gas and oxygen. 10.0 a.m., returned to bed, colour good, blood

pressure 120/75. Slept without moving until 6.0 p.m.; somewhat restless until 8.0 p.m.; received morphine gr. $\frac{1}{8}$ and slept until next morning, when he awoke asking when operation was going to take place.

Soldier aged 39.—Operation: partial gastrectomy. Heart and chest, nil abnormal. Blood pressure 120/65. Weight 10 st. 6 lb. Thin, active man. Night before operation: pot. brom. gr. xxx. 7.45 a.m., morphine gr. $\frac{1}{4}$ with atropine gr. 1/100. 8.0 a.m., paraldehyde 10½ drams in saline 14 ounces per rectum. 8.30 a.m., asleep; blood pressure 100/60. 9.0 a.m., anæsthetised with gas-oxygen-ether, relaxation obtained with very little ether. 10.30 a.m., returned to bed, colour good, pulse 120, blood pressure 90/60. 4.0 p.m., still asleep, colour good, pulse 100, blood pressure 95/70. Slept until 3 a.m. No vomiting. Convalescence uneventful.

Girl aged 8.—Operation: tonsilectomy. Physical examination, nil abnormal. Tall, big child; weight 6 st. 7.45 a.m., morphine gr. $\frac{1}{8}$ with atropine gr. 1/100. 8.0 a.m., paraldehyde 5 drams in saline 7 ounces. Asleep almost immediately after paraldehyde was run in. Pulse 120, soft; respirations shallow. 9.0 a.m., anæsthesia with 50/50 mixture of gas and oxygen; operation performed without event. 9.45 a.m., returned to bed, breathing very shallow, colour poor. Oxygen administered; bad colour without oxygen. Lobeline gr. 3/10 hypodermically—colour and breathing improved. Patient remained asleep until 8.0 a.m. next morning, very drowsy all next day. Uneventful recovery. This child was wrongly graded, and should not have received the morphine.

Boy aged 5½.—Operation: tonsilectomy. Physical examination, nil abnormal. Very fit little boy; weight 2 st. 8 lb. 8.15 a.m., paraldehyde 2 drams in saline 3 ounces. 8.40 a.m., asleep, colour good, pulse 98. 9.0 a.m., anæsthetised—gas and oxygen with very little ether at start only. 9.30 a.m., back in bed, colour good. 10.30 a.m., somewhat restless, condition good. 12 noon, crying, periods of sleep. Seen at 4.0 p.m., awake, condition good, vomited twice.

AVERTIN (TRI-BROMO-METHYL-ALCOHOL).

Avertin is a white crystalline powder which is only soluble 1 in 28 of water. If heated above 40°C. it decomposes and dibromacetaldehyde is formed, which will cause injury to the rectal mucosa. Like paraldehyde, it is administered by the rectum. Immediately before use the solution must be tested to make certain that no decomposition has occurred. For this purpose Congo red 1 : 1,000 solution is recommended by the manufacturers. It should remain orange red. Any trace of blue tint would indicate decomposition, and the solution should be discarded.

Avertin is very quickly absorbed by the intestinal mucous membrane. It is largely excreted through the kidneys, and to a less degree by the liver. It was originally available only in powder form, and the time required for the preparation of the solution was a great disadvantage. The manufacturers now sell a solution which is kept stable by the addition of amylene hydrate. Each c.c. of the solution contains one gram of avertin. From this a 2½ per cent solution is prepared with distilled water at 35°C. Dosage varies from about 0.05 to 0.15 grm. per kilogram body weight, the average dose being about 0.1 grm. per kilo.

Using the classification already described my average dosage was, approximately, as follows: Group 1, 0.05 to 0.08 grm. per kilo; Group 2, 0.1 grm. per kilo; Group 3, 0.12 grm. per kilo; Group 4, 0.12 with the addition of morphine, hyoscine, etc. I have never exceeded a dose of 0.12.

An enema the night before is desirable but not asolutely necessary. Morphine given an hour before operation enables one to obtain narcosis with smaller doses of avertin, but is objected to by some workers on account of respiratory depression.

The solution is very slowly run into the rectum half an hour before operation. Excitement is rare, and the patient very quickly goes to sleep in a natural manner; but narcosis progressively deepens for another ten to fifteen minutes. It is quieter and deeper than that of paraldehyde. The patient is relaxed and will more easily lose his airway. In many ways it reminds one of chloroform anæsthesia.

Contra-indications are few. Disease of the kidneys and

liver, rectal disease, and advanced pulmonary tuberculosis are the chief ones mentioned by German writers; but the patient's condition and the degree of toxæmia present must, of course, be the real guiding factors when deciding for or against the drug.

Recovery of consciousness occurs usually in from one to four hours; the period depends largely upon the amount of morphia and the anæsthetic used. Restlessness is rare. Frequently there supervenes a period during which the patient, though lying quietly with eyes closed, can talk and take drinks, etc., but will afterwards remember nothing of what has happened during this time. Vomiting rarely ensues, but if it does it is not remembered.

Illustrative Cases.

Woman aged 50.—Operation for carcinoma of breast. Heart and lungs, nil abnormal. Weight 8 st. 6 lb.; a small, spare, "wiry" woman. Blood pressure 130/80. 11.0 a.m., morphine gr. 1/5 with atropine gr. 1/100. 11.30 a.m., avertin fluid 5.3 c.c. in distilled water 214 c.c. (0.1 grm. per kilo); solution shaken hard, tested with Congo red, and slowly run into rectum with catheter and funnel. 11.45 a.m., asleep; blood pressure 90/65. 12 noon, anæsthetic (gas and oxygen only), good pulse and colour throughout. 1.10 p.m., blood pressure at end of operation 90/65. 2.30 p.m., patient moved and spoke, but fell asleep again. 4.0 p.m., patient quite conscious, no vomiting. Uneventful recovery.

Man aged 68.—Operation: œsophagoscopy for carcinoma of œsophagus. Lungs, chronic bronchitis. Heart sounds distant and feeble, pulse soft, 88. General condition, wasted and feeble. 11.0 a.m., morphine gr. $\frac{1}{8}$ with atropine gr. 1/100. 11.30 a.m., avertin fluid 5.1 c.c. in distilled water 203 c.c. (0.08 grm. per kilo). 11.40 a.m., asleep. 12 noon, pharynx anæsthetised by cocaine spray; œsophagoscopy performed without general anæsthetic. Patient asleep until 3.0 p.m. Uneventful recovery.

I have, unfortunately, three other cases to report when the patient died. The first two occurred in 1929—using an early sample of avertin which I obtained from Germany.

The third had avertin fluid. The cause of death is not very evident. I am sure the solution of avertin was correctly made and tested; probably the error occurred in gauging the dose, and, as will be seen, two were bad risks. Avertin is said to be excreted as bromine, which, in common with the other halogens, I believe is prone to attack the liver.

Last year I anæsthetised a man of 61 for partial gastrectomy—he had avertin (no morphine) followed by ether. His condition was satisfactory until two days after operation, when he collapsed with signs of pneumonia. The only post-mortem findings were hypostatic pneumonia on both sides, and fatty infiltration of the liver throughout. Something akin to delayed chloroform poisoning may be possible. Combined with morphine the respiration becomes depressed; in fact, the condition of the patient reminds one very much of chloroform anæsthesia—shallow breathing, soft pulse, absolute relaxation.

Man aged 68.—Operation for carcinoma of tongue: insertion of radium needles. Heart, second pulmonary sound accentuated, nil else abnormal. Systolic blood pressure 120. Lungs, chronic bronchitis and emphysema. Weight 10 st. 2 lb. 11.0 a.m., omnopon 1 c.c. (=gr. ⅓), atropine gr. 1/50. 11.30 a.m., avertin 6.0 grams in distilled water 240 c.c. (This is 0.3 grm. less than 0.1 grm. per kilo.) Asleep on arrival at theatre at 12.15. No anæsthetic needed—patient moved once or twice when needles were inserted, but not enough to prevent, or delay, the operation. Systolic blood pressure 70, pulse 90, volume fair. Patient never recovered consciousness. He became cyanosed in spite of the administration of oxygen, and his pulse became more and more feeble, until he finally died at 2 a.m. next day.

It is perhaps hardly fair to blame avertin for death. The man obviously had too much omnopon and avertin, as shown by the fact that he needed no other anæsthetic. There may also have been another factor—obstructed airway. Usually tongue cases are intubated and the tube left in until the patient's reflexes have returned and he will no longer tolerate it. In this case no intubation was done; the patient maintained his own airway whilst in the theatre, and the

house surgeon assured me that there was no evidence of obstruction to breathing afterwards.

Woman aged 27.—Operation: appendicectomy. Heart and lungs, nil abnormal. Weight 11 st. 4 lb. Healthy woman. 12 noon, morphine gr. $\frac{1}{4}$ with atropine gr. 1/100. 12.15 a.m., avertin 7.2 grm. in distilled water 290 c.c. (0.1 grm. per kilo). 12.40 a.m., patient asleep; pulse full and regular; no blood-pressure records taken. Anæsthetic, nitrous oxide-oxygen-ether. It was necessary to give ether virtually the whole time to obtain relaxation. Patient returned from theatre fit, but she never recovered consciousness, her pulse gradually became more and more feeble, and in spite of every restorative measure she finally died at 3.0 p.m. the following day, 26$\frac{3}{4}$ hours after the administration of the avertin. Unfortunately we were unable to obtain a post-mortem examination.

Woman aged 63.—Operation for exophthalmic goitre. Heart, enlarged 4$\frac{1}{2}$ in. to left of mid-sternal line. Pulse on admission 120, on day before operation 84. Blood pressure 138/78. Report from physician-in-charge, electrocardiographic department: "No contra-indication for operation." Basal metabolism rate +46 per cent. Considerable loss of weight. Weight 8 st. 7$\frac{1}{2}$ lb. Typical thyrotoxic patient. 1 p.m., morphine gr. $\frac{1}{8}$ with atropine gr. 1/100. 1.30 p.m., avertin fluid 5.4 c.c. (0.1 c.c. per kilo) in distilled water 218 c.c. 2.0 p.m., patient not asleep on arrival at theatre. Nurse reported some of avertin solution returned; therefore hyoscine gr. 1/100 given hypodermically. Anæsthetic: bilateral paravertebral cervical block, 2 per cent, novocain with adrenalin 1: 400,000. During the operation it was necessary to give a little gas and oxygen to keep patient still. Patient opened eyes and looked round in a dazed way before leaving theatre. Continuous oxygen and rectal saline given (routine) on return to ward. 5 p.m, unconscious, pulse 140, irregular, cardiazol and digitalin given. 8.30 p.m., breathing shallow; pulse 160, irregular. Lobelin CO_2 and O_2 given, also digitalin, with very little effect. Pulse and respiration became feebler and feebler, and patient died at 9.45 p.m. No post-mortem examination.

The Barbiturates.

There are many barbituric acid compounds in use and they vary greatly in toxicity. Two facts which are important with regard to the effects produced by the barbiturates are: (1) The more toxic the compound, the smaller the dose required to produce a hypnotic effect; and the more rapidly is that effect produced. (2) The smaller the dose, the shorter the period during which the drug will act; and the more rapid will be its elimination from the patient's system.

The barbiturates have been used in various forms for many years; they are essentially hypnotic drugs, and only produce anæsthesia in large and dangerous doses. Used as basal hypnotics the optimum dose is comparatively easy to judge, especially when given intravenously; but it is a different matter if the deeper stages approximating to anæsthesia are intended; it is very easy to administer an overdose. Morphine given beforehand increases their action, and smaller doses are needed to produce unconsciousness. As a general rule the systolic blood pressure is lowered some twenty to thirty points after the intravenous administration of a barbiturate, slightly more than this if morphine has been given. In many cases the blood pressure returned to normal within about ten minutes. It can always be raised by the inhalation of a little ether.

The hypnotic state resulting from the administration of a barbiturate has been divided into three stages. These merge one into another, and in the transition either into, or out of, complete unconsciousness a patient may pass through each stage; but absorption or elimination may be so rapid that the stages are indistinguishable. These stages are:—

1. Complete unconsciousness. Only obtained when a dose sufficiently large to be effective has been administered. The corneal reflex is sluggish or lost, but unless considerably more than a hypnotic dose has been administered, the patient will respond to painful stimuli.

2. A stage of semi-consciousness, during which a patient can be roused, will take drinks, and speak, but will lapse

into sleep again when left alone. Some few cases (especially children) become restless in this stage; this may vary from slight movement to great excitement requiring forcible restraint. The patient has no memory whatever of events which have taken place during this period.

3. A period during which the patient is quite conscious, but feels drowsy and will sleep if left alone.

Elimination of Barbiturates.

I am unable to learn anything very definite about the elimination of the barbiturates. Probably they are excreted by the kidneys, after having been broken up by the liver. I gave 4½ grains of nembutal by mouth to a patient who was suffering from toxic jaundice. He fell asleep immediately, and we were able to extract several teeth without an anæsthetic. He remained unconscious for ten hours and drowsy for two days. The probable explanation of this appears to be that his liver was unable to deal with the drug.

An antidote has been stated to be caffein sodium benzoate. I administered this to a woman who had taken a large amount of dial. She was deeply unconscious with no corneal or other reflexes. I gave her caffein 0.5 grm. with sodium benzoate 0.5 grm. intravenously, and although she did not regain consciousness immediately, her corneal reflex returned, and she moved when pricked with a needle immediately afterwards. Thyroxin, by raising the metabolic rate, is also said to hasten elimination. Zerfas reports that animals given fatal doses of sodium amytal died of respiratory failure which usually precedes that of the circulatory system, though failure of both sometimes occurs simultaneously—particularly if the drug is injected rapidly.*

Administration of Barbiturates for Nasal Narcosis.

The three barbituric compounds which have lately been employed as basal narcotics are sodium amytal (sodium iso-amyl-ethyl-barbiturate), nembutal (sodium ethyl—(1 methyl-butyl)—barbiturate), and pernocton (sodium salt of secondary butyl-β-brom. allyl barbituric acid). They are all quickly acting drugs and very toxic, but nembutal and pernocton

* *British Medical Journal*, November 29th, 1930, p. 897.

are twice as toxic, and therefore twice as powerful, as sodium amytal. The latter must be administered in larger doses and the recovery period is therefore longer. Nembutal and pernocton are said to be equal in toxicity, and I think that is borne out by their recovery periods, which are approximately equal.

Pernocton is supplied ready for injection in 10 per cent solution, which saves time; but in my experience it is neither so easy to use, nor so pleasant for the patient as nembutal. I have only given it intravenously, and the small bulk makes the dose difficult to judge—several of the cases vomited during both administration and recovery periods, and in several excitment was present during both periods. With nembutal, I have seen virtually no ill effects, apart from one case of excitement during recovery. This occurs in a small proportion of adults and is seldom extreme, but it is much commoner with children, especially when administered by mouth.

Whenever possible it is preferable to give the barbiturates intravenously. They may, however, be administered intramuscularly, or by the mouth or rectum. By these routes their action is delayed and the correct dose is more difficult to judge. Intravenous administration is easy, and the minimal effective dose—just enough to produce unconsciousness, and no more—which is desirable can be gauged with accuracy.

Amytal powder is supplied in 1.0 grm. quantities and nembutal in 0.5 grm. quantities, sterilised in ampoules. Immediately before use solutions are prepared by dissolving the powder in 10 c.c. of sterilised distilled water. Amytal is therefore administered in 10 per cent and nembutal in 5 per cent solution. Pernocton, which is more stable, is supplied in 10 per cent solution in ampoules containing 5 c.c. The solution is run into the vein very slowly (1 c.c. per minute), meanwhile the patient is engaged in conversation. As the injection proceeds his speech becomes more and more slurred, until finally he fails to answer questions, and sinks into a deep sleep. The administration is stopped immediately he loses consciousness. In this way correct dosage is ensured.

If a small dose of morphine and hyoscine be given beforehand, the intravenous injection is often not remembered at all. Usually very little anæsthetic is needed, although patients vary considerably in this respect; but even after ether vomiting is virtually unknown. Recovery, as a rule, takes place in from two to six hours, followed by the protracted period of drowsiness already mentioned, which may last as long as twenty-four hours.

ILLUSTRATIVE CASE.

Man aged 56.—Operation: partial gastrectomy. Heart and lungs, nil abnormal. Blood pressure 140/80. Fit man of 11 st. 5 lb. 9 a.m., hyoscine compound "A" (hyoscine hydrobrom. gr. 1/100, morphine sulph. gr. $\frac{1}{8}$, atropine sulph. gr. 1/180). 9.50 a.m., nembutal 7 c.c. of 5 per cent solution in sterile distilled water intravenously. Patient went to sleep in midst of a sentence. No excitement. 10.0 a.m., anæsthetic, nitrous oxide and oxygen with very little ether; good relaxation very easily obtained. Blood pressure 115/60. 11.25 a.m., returned to bed. Blood pressure 110/60. Good colour, breathing normally. 2.15 p.m., recovered consciousness. Pulse good. Slept until 4.0 p.m. without morphine. Final recovery uneventful.

I have no deaths or mishaps to record with the barbiturates. The only case in which they were blamed (I feel sure unjustly) for subsequent events was this:

A girl of 16, apparently healthy, but with a history that she had had several faints and that her fingers often went blue, had an operation for appendicitis. She was given atropine gr. 1/100 and 5 c.c. of a 5 per cent solution of nembutal intravenously before operation. No morphine. The anæsthetic was open ether. The operation period was normal and uneventful. While the dressings were being applied, and some minutes after the ether administration had ceased, she quite suddenly stopped breathing and her pulse became impalpable. It was only after the most energetic restorative measures that she started to breathe and her pulse returned. Subsequently she had about forty convulsive fits, probably the result of failure of her cerebral circulation. She did not

regain full consciousness for two days, but she finally recovered completely.

I have no idea what caused this patient's collapse—her pulse was good and she behaved in a perfectly normal manner before and during the operation; but I feel sure that whatever the cause it cannot be justly attributed to the nembutal she had had.

Conclusions.

I must confess a personal preference for either paraldehyde or nembutal. For children I like the former because the barbiturates must generally be given to them by mouth or rectum; and thus administered they so often cause restlessness. For adults nembutal gives equally good results, and is less worrying to the patient than paraldehyde. Post-operative restlessness when present is usually slight, and may be controlled by morphine or rectal paraldehyde. I think that both paraldehyde and nembutal are eminently safe in expert hands.

Combinations of drugs act more powerfully, and smaller doses are needed than of any one drug alone. Elimination is easier and quicker than saturation with one. A little morphine, just sufficient nembutal or paraldehyde to produce sleep, and then nitrous oxide and oxygen or ethylene, with a minimum of ether, if necessary, to produce relaxation, give the patient most protection, least injury, and an easy awakening.

Basal narcosis marks a distinct advance in the administration of anæsthetics. The advantages of minimised mental shock are sufficiently great in themselves to warrant its use; but since, in addition, it enables us to ensure for our patients absence of vomiting and a post-operative period of unconsciousness, during which most of the anæsthetic is eliminated, and a great deal of pain evaded, its use is doubly justified. The public are beginning to recognise its merits, and I feel sure that before long the demand for it will be general.

A certain amount of experience is necessary before one can hope to obtain consistently good results. The adminis-

tration of an anæsthetic becomes a very different procedure from the older methods we have been accustomed to, and it will be a great pity if reproach is brought upon basal narcosis by the inexpert.

REVIEW.

Elementary and Advanced Anæsthesia. By Dr. K. B. MADAN. *Lecturer in Anæsthetics, King Edward Medical College, Lahore,* Illustrated, price 5 Rupees.

This little book is noteworthy as being the first of its kind produced in India. Indeed its author is a pioneer in being the first to hold a lectureship in Anæsthetics in India. It is good to see that the almost complete monopoly which chloroform held for so many years in practice within our Indian Empire is giving way before the spreading of knowledge of the principles of anæsthetic practice. Judging from the contents of the book, that knowledge is well up to date on the part of the instructors in anæsthetics, for avertin and percaine come in for consideration, although the latter certainly receives less than its merits demand, and the most recent barbiturates are not yet within the author's ken. The book has many minor faults of language and of arrangement, and we wish that the author had given more space to results of his own experience and less to the writings of others; but nevertheless he is to be congratulated on his industry and on producing a work which will doubtless be of great value in the country for which it is intended.

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